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Abstracts

76th Meeting of the Ulster Society of Internal Medicine, Friday 20th October 2006

The Mater Hospital Trust, Belfast

PROGRAMME:

2.00pm Welcome - Chairman: Dr David Higginson
2.00pm Spoken Presentations I
3.10pm Invited Abstract: Cardio-Renal-Anaemia: Prevalent and treatable. Dr Damian Fogarty, Senior Lecturer and Consultant Nephrologist, Belfast City Hospital Trust.
3.40pm Afternoon Tea
4.10pm Spoken Presentations II
4.35pm Invited case from The Mater Hospital Trust
4.50pm Presentation of prize for best abstract
5.00pm Close

PRESENTED ABSTRACTS

Aquagenic wrinkling of the palms in adult patients with cystic fibrosis.

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Aquagenic Wrinkling of the Palms (AWP) is characterised by the development of oedematous white plaques on the hands following exposure to water. To date 30 cases of AWP have been reported, 17 of which are in patients with cystic fibrosis (CF)[1]. Only two cases have been reported in males[1,2]. We have identified 45 patients out of a cohort of 102 adult CF patients who describe AWP. 24 cases are male and 21 female. 25 patients give a very good history of wrinkling of the hands when immersed in water, 13 give an average history and formal testing has been carried out in 7 patients with positive results. In most patients the whole of the hand is affected and not just the finger tips. Eight patients state that the reaction is painful. Eight of the patients report desquamation, especially at the finger tips, shortly after the hands are out of water. A similar reaction involving the feet is also described by seven patients. Fourteen describe mild to moderate hyperhidrosis of the hands. We tested seven patients by placing one hand in warm tap water; the other hand was used as comparison. After 15 minutes of immersion AWP was demonstrated in all cases. The whole of the hand appeared white in colour, oedematous and excessively wrinkled.

Conclusion: we present the clinical features of this interesting phenomenon and postulate that the incidence of AWP is likely to be much higher than the small numbers reported in the literature to date.

1. Katz KA, Yan Ac, Turner ML. Aquagenic wrinkling of the palms in patients with cystic fibrosis homozygous for the ΔF508 CFTR mutation. *Arch Dermatol* 2005;**141:**621-624
2. Neri I, Bianchi F, Patrizi A. Transient aquagenic palmar hyperwrinkling: the first instance reported in a young boy. *Pediatr Dermatol* 2006;**23(1):**39-42

Mortality following Percutaneous Endoscopic Gastrostomy: results of the National Confidential Enquiry into Patient Outcome and Death

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Background: Percutaneous endoscopic gastrostomy (PEG) is an accepted method of placing a feeding tube to enable enteral feeding in patients with swallowing difficulties. However the factors associated with morbidity and mortality following PEG have not been studied in detail. We describe the largest audit of mortality following PEG tube insertion.

Methods: Deaths occurring within 30 days following PEG tube insertion in the United Kingdom between April 2002 and March 2003 were identified and a questionnaire sent to the consultant endoscopist for completion.

Results: 719 patients (391 male, median age 80 yrs.; range 26-98 yrs.) who died within 30 days following PEG insertion were identified. 97% of patients had co-existent neurological disease. PEG tubes were inserted by specialised GI physicians in 522 cases (73%). Seventy-two patients (10%) required reversal agents following sedation. Following PEG tube insertion 309 patients (43%) died within one week. Death was due to cardiovascular disease (n=175), respiratory disease (n=508), central nervous system disease (n=358), renal disease (n=38) and hepatic failure (n=11). In 136 cases (19%) the NCEPOD expert panel regarded the procedure as futile.

Conclusions: Mortality and morbidity following PEG tube insertion is not insignificant. Selection of patients is paramount to good patient outcomes. Multi-disciplinary team assessment should be performed on all patients being referred for PEG tube insertion. Attention to pre-procedural baseline

investigations may also reduce morbidity and mortality.

Elevated troponin levels in acute stroke are negatively associated with outcome at 3 months.

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Introduction: It has been suggested that raised troponin levels are associated with unfavourable outcome after acute stroke. The aim of this study was to determine the prevalence of raised troponin T levels (TnT) in a prospective cohort of patients with acute stroke and examine the relationship between TnT and outcome at 3 months.

Methods: Patients with acute stroke were assessed (including National Institutes of Health Stroke Scale, NIHSS) and had TnT measured within 72h. Outcome at 3 months was assessed using modified Rankin scale (mRs). Comparisons between troponin positive and negative groups were made using either students t-test, mann whitney u, or chi-square depending on the characteristics of each variable.

Results: 132 patients were recruited. 3 month follow-up data is currently available on 98. Elevated TnT levels were present in 14 (10.6%). Patients with raised TnT were older (mean [SD] age, 79.2 [8.68] v 72.0 [11.89], p=0.013) and had more severe strokes (median [range] NIHSS 8.5[1-19] v 4[0-25], p=0.04). At 3 months, mRs was significantly higher in the TnT positive group (median [range] 6[1-6] v 1[0-6], p=0.01), as was mortality (50% v 12%, p<0.001). In multivariate analysis, raised TnT (r= 0.27 p=0.01), age (r=0.20, p= 0.01) and NIHSS (r=0.55, p= <0.001) were independently associated with 3 month mRs.

Conclusions: In this prospective study, raised TnT levels were present in 1 in 10 acute stroke patients and were negatively associated with outcome at 3 months. Further studies are required to determine the causes of poor outcome in troponin positive patients.

Contemporory management of acute coronary syndromes: insights from the Mater hospital cardiology database

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Background: Management of acute coronary syndrome (ACS) has changed significantly in recent years. Current guidelines advise early inpatient coronary angiography for many patients. We reviewed the Mater Hospital cardiology database to determine the impact of these guidelines on the management of our patient population and the hospital outcomes in these patients.

Method and Results: Data for all patients admitted to the Mater Hospital, Cardiology Unit with ACS is entered into a database. This was analysed for the period April 2005 to September 2006. Of 598 patients with acute coronary syndrome, 100 (17%) had ST elevation myocardial infarction (STEMI), 133 (22%) had NON-STEMI and 365 (61%) had unstable angina (USA). Seventy-four of 100 (74%) STEMI patients received thrombolytic agents. Sixty of 100 (60%) STEMI patients had coronary angiography (CA) pre-discharge which resulted in revascularisation in 43 (43%), 35 percutaneous coronary intervention (PCI), 8 Coronary artery Bypass surgery (CABG). Fifty-eight of 133 (43%) NON-STEMI patients had CA which resulted in revascularisation in 34 (26%) (26 PCI, 8 CABG). Eighty-seven of 365 (24%) USA patients underwent CA followed by revascularisation 42 (12%) (34 PCI AND 8 CABG). Only 7 of 100 (7%) STEMI patients and 9/133 (8%) NON-STEMI patients underwent exercise treadmill testing prior to discharge. Rates of prescription of evidence-based secondary prevention medications for the whole study population were: aspirin 502/598 (84%), clopidogrel 434/598 (73%), beta-blocker 454/598 (76%), ACE-inhibitor or angiotensin receptor blocker 421/598 (71%) and statin 521/598 (87%). In-hospital mortality rates were 10/100 (10%) for STEMI, 3/133 (2.3%) for NSTEMI and 3/365 (0.8%) for USA patients. The mean (median) length of hospitalisation in days was 13.2 (6) for STEMI, 12.6 (6) for NSTEMI and 6.4 (3.5) for unstable angina.

Conclusions: In contemporary N. Ireland practice, mortality for unselected patients with STEMI remains significant. A large proportion of ACS patients, and particularly those with STEMI and NON-STEMI, have inpatient coronary angiography and subsequent revascularisation procedures. Consequently, few STEMI and NON-STEMI patients currently undergo exercise treadmill testing. The duration of hospitalisation for acute MI probably reflects delays before CA and revascularisation procedures can be performed.

Pseudo-hyperkalaemia and aetiology of thrombocytosis: a six-year retrospective correlation study

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Pseudohyperkalaemia is a rarely encountered event among patients with thrombocytosis. Typically, a raised serum potassium level is observed in the absence of renal failure, and it causes unnecessary anxiety among clinicians. Pseudohyperkalemia may lead to inappropriate administration of calcium resornium, insulin in 5% dextrose in an attempt to lower potassium level. The association between pseudohyperkalaemia and aetiology of thrombocytosis is unclear.

A six-year retrospective audit was conducted on 90 patients with thrombocytosis referred to the Haematology Department in Ulster Hospital Dundonald, a large district general hospital. Over two thirds of this study population had myeloproliferative disorders, and the most common diagnosis was primary thrombocythaemia (39%, n=35). In contrast, reactive thrombocytosis was observed in approximately one third of the cases (31%, n=28).

Pseudohyperkalaemia with apparent potassium level above the upper limit of the normal range (K>5 mmol/l) was observed in the majority of patients with thrombocytosis from any aetiology (66.6%, n=60). The likelihood of finding pseudohyperkalaemia was highest among patients with primary thrombocythaemia (91.4%, n=32/35), followed by polythaemia rubra vera (56%, n=9/16), and reactive

thrombocytosis (32%, n=9/28). A significant positive correlation was observed between the platelet counts and the serum potassium level (Spearman's correlation coefficient, R=0.28, p=0.01).

The falsely raised potassium level is due to the release of intracellular potassium from platelets during clot formation in the specimen bottle (serum sample). This is a time- dependent phenomenon; the use of plasma sample in either Li-heparin or Na-heparin bottles will circumvent this phenomenon.

Systematic review: blood pressure lowering in patients without prior cerebrovascular disease for prevention of cognitive impairment and dementia

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Hypertension and cognitive impairment are prevalent in older people. Hypertension is a known risk factor for vascular dementia (VaD) and recent studies suggest hypertension impacts upon prevalence of Alzheimer's Disease (AD). The aim of this Cochrane review was to determine if treatment of hypertension lowers the rate of cognitive decline and dementia in patients without known cerebrovascular disease.

Trials included in the systematic review were randomized, double-blind, placebo-controlled trails, in which interventions to lower blood pressure (BP) were administered for ≥ 6 months to hypertensive patients. The Cochrane Dementia and Cognitive Impairment Group Specialized Register was searched from 1966-2005 to identify suitable trials. Primary outcomes were incidence of dementia and cognitive change from baseline.

Three trials were identified with 12,091 hypertensive subjects (SHEP 1991, Syst-Eur 1997 and SCOPE 2003). Analysis was performed on the combined results of all three trials.

Results of incidence of dementia outcome: There was a trend towards a significant benefit of treatment (O.R. 0.89, 95% CI 0.69, 1.16). Only Syst-Eur reached significance with results indicating a benefit.

Results of cognitive change from baseline outcome: No significant benefit from treatment (WMD=0.10, 95% CI -0.03, 0.23).

Conclusion: there was no convincing evidence that BP lowering prevents dementia or cognitive impairment in hypertensive patients without prior cerebrovascular disease. There were problems analysing the data, however, due to heterogeneity across trials, number of placebo patients given active treatment and loss to follow-up. Further placebo-controlled trails would be necessary but this raises ethical issues.

An unusual cause of abnormal thyroid function tests

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Abnormal thyroid function test results frequently pose diagnostic challenges. We report a 23 year old female who presented with fatigue. Free T_4 was 28.9pmol/L (7.6-19.7) and TSH 2.06mU/L (0.45-4.5). She had no other symptoms or signs of hyperthyroidism and on examination was clinically euthyroid with no obvious goitre. Interfering antibodies were excluded, sex hormone binding globulin (SHBG) was not elevated (18nmol/L [25-90]) and α-glycoprotein subunit was normal (<0.3IU/L). Following TRH stimulation (Protirelin 200μg), TSH rose appropriately from 1.59mU/L at baseline to 21.1mU/L at 20 minutes. The pituitary was normal on CT imaging.

Sequencing of the thyroid hormone receptor (TR) β gene, on chromosome 3, revealed a point mutation at position 460 with substitution of the normal glutamic acid by lysine (E460K), thus confirming the diagnosis of resistance to thyroid hormone (RTH). Her father and sister were found to share both the biochemical phenotype and the genetic defect, while her brother did not. Free T_4 index (6.0-10.5) and TSH (0.4-3.6mU/L), respectively, were as follows; father, 16.7 and 2.5; sister, 15.8 and 2.9; brother, 9.5 and 4.4. Affected subject also had high serum levels of T_3 and reverse T_3.

RTH is a condition of impaired tissue responsiveness to thyroid hormone. The more common forms are caused by TRβ gene mutations and inherited in a dominant fashion. Of the 170 different TRβ mutations, E460K has been identified in three other families. This mutation reduces the affinity of TRβ for T_3 to 25% that of the normal receptor. RTH should be considered in patients presenting with elevated FT_4 and unsuppressed TSH. Its identification prevents unnecessary investigation and inappropriate treatment in the patient and in other affected family members.